	Unadjusted population			PSM population		
	Bivalirudin N = 4370	UFH N = 19,564	p	Bivalirudin N = 3649	UFH N = 3649	p
	%	%		%	%	
Death	0.3	1.0	<0.0001	0.3	0.7	0.01
Stroke	0.1	0.3	0.054	0.2	0.3	0.22
MI	0.6	1.2	0.0004	0.5	0.8	0.25
Amputation	1.3	4.5	<0.0001	1.5	2.0	0.09
Transfusion	3.4	10.0	<0.0001	4.0	5.3	0.009
MACE	2.2	6.6	<0.0001	2.3	3.5	0.003
NACE	5.4	14.8	<0.0001	5.9	7.9	0.0009

Conclusions: This analysis from a very large US hospital database suggests that the use of bivalirudin anticoagulation for PAI may confer significant clinical benefits over heparin. These results require confirmation in a prospective randomized trial.

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Bivalirudin versus Heparin for Percutaneous Coronary Intervention: An Updated Meta-Analysis of Randomized Controlled Trials

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Background: Controversy exists regarding the optimal choice of anticoagulation regimen for percutaneous coronary intervention (PCI). We performed a meta-analysis of randomized controlled trials (RCT) to compare bivalirudin (bival) versus heparin with provisional or routine glycoprotein IIb/IIIa inhibitor (GPI) use on 30-day outcomes following PCI.
Methods: Medline/Pubmed and Cochrane CENTRAL were searched along with recent abstract presentations at national meetings for all RCTs comparing BIV with provisional GPI use versus heparin with provisional or routine GPI use for PCI. Pooled estimates of 30 day outcomes were generated for with random-effect models to compare the treatment groups. Data is presented as odds ratios (OR) [95% confidence intervals].
Results: Our analysis included 14 studies with 30,446 patients that were randomized to either bivalirudin with provisional GPI use (n=14,869) or heparin with provisional GPI use (n=6,451) or heparin with routine GPI use (n=9,126). There was no significant difference between anticoagulation with bival compared with heparin for 30 day death (OR 0.94 [0.78-1.14]) or myocardial infarction (OR 1.11 [0.97-1.27]). Early stent thrombosis was significantly greater with bivalirudin compared with heparin (OR 1.62 [1.18-2.23], p=0.003), especially when comparing bivalirudin versus heparin with provisional GPI use (OR 2.09 [1.26-3.47], p=0.005) or among STEMI patients (OR 2.17 [1.15-4.10], p=0.02). However, bivalirudin reduced the risk of major bleed (OR 0.58 [0.49-0.69], p< 0.0001) and TIMI major bleeding (OR 0.58 [0.47-0.71], p< 0.0001) compared with heparin. Meta-regression analysis demonstrated that bleeding risk with use of heparin significantly increases with increasing GPI use (p=0.02).
Conclusions: Meta-analysis of 14 RCTs with 30,446 patients demonstrated that bivalirudin is associated with higher risk of stent thrombosis but lower risk of major bleeding compared with heparin.

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Predictors Of Stent Thrombosis After Primary Percutaneous Coronary Intervention And Risk for 30-Day Mortality: Analysis from the HORIZONS-AMI and EUROMAX trials

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Background: The risk of early (≤30 day) stent thrombosis (ST) is considerable after primary PCI for STEMI. We sought to determine the independent predictors of early ST and evaluate the risk of mortality after ST according to antithrombotic therapy used during the index primary PCI.
Methods: In a patient-level pooled analysis from the HORIZONS-AMI and EUROMAX trials, we studied 5,800 patients undergoing primary PCI at 188 sites, randomized to either bivalirudin or heparin ± a glycoprotein IIb/IIIa inhibitor (GPI). Predictors of ST were determined by multivariate logistic regression, and 30-day mortality was evaluated according to timing of ST and antithrombotic treatment received.
Results: Of 100 patients (1.7%) who developed early ST, 20 (20%) died within 30 days of enrollment. By logistic regression, independent predictors of early ST were pre-PCI TIMI grade flow 0-1 and Killip class ≥2 at presentation. Bivalirudin was associated with higher rates of early ST (2.1% vs. 1.4%, RR=1.51, adj. p-value=0.07) driven by a higher incidence of acute ST (1.2% vs. 0.2%, RR=6.04, p< 0.0001) with similar rates of subacute ST (0.9% vs. 1.2%, RR=0.74, p=0.24) in comparison to heparin ± GPI. However, 30-day mortality rates among patients with ST were lower in the bivalirudin-treated subset; this was consistent for both acute and subacute ST (Table). As a result, only 4/2,889 bivalirudin-treated patients died within 30 days after early ST compared to 16/2,911 heparin ± GPI treated patients (0.14% vs. 0.56% respectively, P=0.01).
Conclusions: Killip class ≥2 during acute MI presentation and pre-procedure TIMI grade flow 0-1 are independent predictors of early ST after primary PCI. Although the risk of ST within 30 days is higher among patients treated with bivalirudin due to a greater hazard of acute ST, death attributable to early ST is substantially less common in patients having received bivalirudin compared to heparin ± GPI.

30-day Mortality in Patients with Early ST

Timing of ST	Bivalirudin (n=2,889)	Heparin ± GPI (n=2,911)	Relative risk [95% CI]	P-Value*
Acute (≤24 hrs)	1/36 (2.8%)	1/6 (16.7%)	0.17 [0.01, 2.16]	0.14
Subacute (1-30d)	3/25 (12.0%)	15/34 (44.1%)	0.29 [0.10, 0.88]	0.01
Early (≤30d)	4/60 (6.7%)	16/40 (40.0%)	0.19 [0.07, 0.52]	0.0002

*Cochran-Mantel-Haenszel χ^2 test stratified by study

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Association Of Activated Clotting Times During Percutaneous Coronary Intervention and Clinical Outcomes

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Background: Monitoring the intensity of anticoagulation with heparin during percutaneous coronary intervention (PCI) using the activated clotting time (ACT) is one of the most frequently used tests in invasive cardiology. However, despite its ubiquitous use, controversy remains regarding the association of ACT with ischemic and bleeding events.
Methods: We reviewed all PCI procedures performed at Mayo Clinic (Rochester, MN) between 2001 - 2012 and evaluated the association between the ACT value at the time of PCI and in-hospital and 1-year outcomes. For descriptive purposes, ACT values were grouped into tertiles. We used logistic and Cox proportional hazards regression models to estimate the association of ACT, modeled continuously, with outcomes while accounting for baseline characteristics.
Results: Of 12,059 patients studied, 3,978 (33.0%) had ACT < 227, 4,047 (33.6%) had ACT 227-285, and 4,034 (33.4%) had ACT >285. Groups were similar regarding baseline and procedural characteristics. In univariate analysis, ACT had associations with in-hospital and 1-year clinical events; however, after multivariable adjustment, ACT at the time of device activation was not independently associated with outcomes (Table).

Association Between Activated Clotting Time (per 50 sec increase) and Clinical Outcome

	Unadjusted OR (95% CI)	p value	Adjusted OR (95% CI)	p value
In hospital overt bleeding	0.94 (0.88, 1.01)	0.09	1.00 (0.93, 1.08)	0.96
In hospital death	0.79 (0.71, 0.88)	<0.0001	1.01 (0.89, 1.15)	0.85
In hospital death/MI	0.98 (0.93, 1.04)	0.51	1.01 (0.95, 1.08)	0.77
1 year cardiac death/MI	0.97 (0.93, 1.01)	0.16	1.00 (0.95, 1.04)	0.81
1 year cardiac death/MI/TLR	0.97 (0.94, 1.00)	0.07	0.99 (0.96, 1.03)	0.57

Abbreviations: MI, myocardial infarction; TLR, target lesion revascularization
Models adjusted for age, sex, body mass index, hypertension, diabetes, heart failure, smoking, cholesterol level, prior PCI/CABG, shock at presentation, MI at presentation, stent type (drug eluting vs bare metal), glycoprotein IIb/IIIa use